Diagnosis of Brugada Syndrome During Fever: A Case Report from a Tertiary Care Centre

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Abstract

A 19 yr old boy came to our hospital with high grade fever since two days. We made clinical impression of malaria. Routine ECG showed rsr pattern and ST elevation in V1, V2. After excluding all other conditions and retrospective counselling, we made diagnosis of Brugada syndrome. Screening baseline ECG of his family members is positive for Brugada ECG pattern. Echocardiogram is normal. Since patient is asymptomatic, we referred him to higher cardiac centre for electrophysiological testing.

Case Report

A 19 yr old boy came to our hospital with high grade fever since two days. Physical examination revealed mild splenomegaly. We admitted him in casualty with clinical impression of malaria and empirically treatment started with antimalarial drugs, antibiotics, and antipyretics. Investigations revealed TLC (total leucocyte count) 6500/cc, Hb 13.5 gm/dl, smear for malarial parasite-negative, complete urine examination-normal. On third day of admission, patient was febrile (Temperature: 103°F), toxic, hence ECG was advised to see any changes of myocarditis. ECG findings include sinus tachycardia, incomplete RBBB and ST elevation > 2 mm in V1, V2. Cardiac examination revealed tachycardia but no gallop. Patient had no chest pain/discomfort/heaviness.

Clinically, biochemically and ECG wise, we excluded the possibility of acute coronary syndrome, myocarditis, pericarditis, early repolarisation. Biochemical tests including cardiac enzymes and electrolytes were within normal limits and the chest X-ray was unremarkable. Then we suspected the possibility of Brugada ECG pattern (Figure 1). On retrospective counselling he doesn’t have had syncopal attacks or paroxysmal palpitations. His father had a sudden death at home during night at the age of 25 yrs and cause was not ascertained. Patient’s grandfather had sudden death at home at the age of 50 yrs who had no pre morbidities (Figure 2). Patient has two younger brothers, both are asymptomatic. With this history and ECG findings, we made...
diagnosis of Brugada syndrome (diagnostic criteria: Table 1) and referred to cardiologist. Cardiologist did Echocardiogram which revealed no evidence of structural heart disease. Cardiologist also made same diagnosis and referred back to physician for control of pyrexia. We took screening ECG for both brothers and one sibling has similar Brugada pattern (type2) in V1, V2 (Figure 3) and other sibling has doubtful Brugada pattern (type3) in V1 only (Figure 4). Patient’s ECG was taken after his recovery from fever which also showed spontaneous Brugada pattern (type1) but amplitude of ST elevation is decreased (Figure 5). Since patient is asymptomatic, as per protocols, we referred him and his brothers who are also asymptomatic, to go to higher cardiac centre for drug challenge and electrophysiological testing.

**Discussion**

Brugada syndrome is a heritable arrhythmogenic disease, with an autosomal dominant mode of transmission, with male preponderance (M:F 9:1). The first case of brugada syndrome was reported in 2006 in India.

Majority of the affected individuals are south East...
Acute coronary syndrome, electrolyte imbalance, testing should be considered. If drug challenge is positive, electrophysiological (EP) and Type 1 ST segment elevation in family members. Family history of sudden cardiac death at < 45 years main indication for drug challenge is in patients with regardless of the results of drug testing. Thus, the Most such symptomatic patients will receive an ICD tachyarrhythmia, or nocturnal agonal respiration. Have documented VF, self-terminating polymorphic that drug challenge is not necessary in patients who Brugada pattern on the ECG, including overdoses with neuroleptic drugs or cyclic antidepressants. Brugada pattern in ECG. Premature inactivation of the sodium channel increases with higher temperature in some SCN5A mutations, suggesting that febrile states may unmask Brugada pattern or increase the risk of arrhythmias. One retrospective analysis of 111 patients with BrS suggest that fever is a trigger for ECG changes and cardiac arrest. Drugs that block cardiac sodium channels produce a transient Brugada pattern on the ECG, including overdoses with neuroleptic drugs or cyclic antidepressants.

ECG abnormality is the hallmark of Brugada syndrome but fluctuations in the ECG pattern appear to be common in BrS. Hence both ECG and clinical features are important for making diagnosis of BrS.

Patients with Brugada type 2 or type 3 ECG pattern, occasionally type1 should undergo drug challenge test with sodium channel blockers (e.g., flecainide, procainamide, ajmaline, pilsicainide) to unmask ECG findings since only type1 pattern is considered as a diagnostic criterion. However, not all patients with a type 2 or type 3 Brugada ECG need to undergo drug challenge. In particular, we suggest that drug challenge is not necessary in patients who have documented VF, self-terminating polymorphic VT, unexplained syncope strongly suggestive of a tachyarrhythmia, or nocturnal agonal respiration. Most such symptomatic patients will receive an ICD regardless of the results of drug testing. Thus, the main indication for drug challenge is in patients with family history of sudden cardiac death at < 45 years and Type 1 ST segment elevation in family members. If drug challenge is positive, electrophysiological (EP) testing should be considered.

Importantly, some of the conditions such as acute coronary syndrome, electrolyte imbalance, myocarditis, pericarditis, early repolarisation, drug over dosage, ARVD should be carefully excluded during the differential diagnosis.

Risk factors for sudden cardiac arrest are, presence of symptoms before diagnosis, a spontaneous type 1 Brugada pattern in ECG, the inducible ventricular arrhythmias during the electrophysiological testing and male sex.

Patients with BrS have very poor prognosis if left untreated because they are prone for development of polymorphic VT and sudden cardiac death.

No drug is efficient to prevent SCD. The only therapy with proven efficacy in preventing sudden death is an implantable cardioverter-defibrillator (ICD) implantation. 10-year follow-up after ICD implantation has been associated with 0% mortality.

Conclusion

Brugada syndrome is a familial channelopathy affecting predominantly males. Our case report supports the effect of temperature on ECG changes of BrS and all physicians should be aware of this fluctuating ECG changes as one of the characteristic ECG abnormality. Brugada syndrome should be kept in differential diagnosis of incomplete RBBB with ST elevation in V1, V2 when all other factors have excluded. All the people between the age group of 15-45 yrs who had unexplained syncpe or family history of sudden death < 45 yrs for which cause was not ascertained, should consult physician for routine baseline ECG and all family members of patient with BrS should have screening ECG. ICD implantation is the only proved, effective treatment for BrS.

References

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